JOURNAL OF COMMUNICATION

Journal of Communication ISSN 0021-9916

ORIGINAL ARTICLE

Alexithymia and Impairment of Decoding Positive Affect: An fMRI Study

Colin Hesse[1], Kory Floyd[2], Emily A. Rauscher[3], Nick E. Frye-Cox[4], John P. Hegarty II[5], & Huiling Peng[6]

1 Department of Communication Studies, University of Missouri Columbia, Columbia, MO, 65203, USA
2 Department of Communication, Arizona State University, Tempe, AZ, 85287, USA
3 Department of Communication, University of Southern Indiana, Evansville, IN, 47712, USA
4 Department of Human Development and Family Studies, University of Missouri Columbia, Columbia, MO, 65211, USA
5 Department of Radiology, University of Missouri Columbia, Columbia, MO, 65211, USA
6 Department of Psychological Sciences, University of Missouri Columbia, Columbia, MO, 65211, USA

Previous research has implicated alexithymia as a psychological impairment to accurately decode emotional messages. This study attempted to explore potential neurological reasons for this impairment. Using functional brain imaging procedures, an experimental design was undertaken to assess group differences between individuals high and low in alexithymia on brain activation while viewing images of individuals displaying neutral or positive affect. While controlling for activation due to neutral affect images, results showed less activation for alexithymic versus nonalexithymic individuals due to positive affect images in several areas of the brain, including the amygdala and the hippocampus. Several implications and directions for future research are also discussed.

doi:10.1111/jcom.12039

A formidable corpus of theory links affective decoding skill to biopsychosocial health. From the vantage of evolutionary theory, the ability to interpret and respond accurately to the emotional expressions of conspecifics is clearly adaptive (Floyd, 2006), as such expressions can signal both opportunities and dangers that have implications for survival and reproduction (see Darwin, 1872; Ekman, 2003). Accurately interpreting an expression of affection, for instance, provides one an opportunity to form or reaffirm a close relationship—which can be important for both survival and reproduction—that is missed by failing to interpret that expression accurately.

In the communication discipline, theories such as communication accommodation theory (Giles, 2008) and affection exchange theory (Floyd, 2006) claim that

Corresponding author: Colin Hesse; e-mail: hessecr@missouri.edu

accurate decoding of emotions is essential for healthy social interaction. Empirical evidence supports that claim. For example, skill at decoding positive and negative emotions is linked to satisfaction in close relationships (Carton, Kessler, & Pape, 1999). Increases in stress lead to decreases in decoding ability (Keeley-Dyreson, Bailey, & Burgoon, 1991). Multiple studies of patient-provider communication document the benefits of physicians' affective decoding ability to patients' health (DiMatteo, Hays, & Prince, 1999). It is apparent that individuals better able to decode emotions are advantaged in terms of overall wellness.

An evolutionary perspective predicts that the body evolves in response to evolutionary pressures. That is, natural selection creates and refines physical features that are adaptive, meaning that they serve functions related to survival and/or reproduction (see Williams, 1996). To the extent that emotion-decoding ability is adaptive, natural selection pressures would lead to the evolution of physical features that enhance that ability. Indeed, advances in neuroscience and neuroimaging techniques have pointed to specific neural structures—including the amygdala and the superior temporal gyrus—as the facilitators of emotion decoding (see Egolf, 2012).

Inherent in an evolutionary explanation is the expectation of individual variation, however. Natural selection provides that some members of a species are better prepared than others to meet the evolutionary demands of their environments (Darwin, 1859). A relevant question, therefore, is what accounts for the variance in an adaptive ability, such as emotion-decoding ability. One feature that accounts for variation is the psychological trait of alexithymia, which impairs both the experience and the expression of emotion (Taylor, Bagby, & Parker, 1997). Indeed, alexithymia appears to lead to a host of communicative deficits, including nonverbal immediacy, affectionate communication, and relational messages of intimacy (Hesse & Floyd, 2008, 2011). Still, scholars have yet to understand why exactly alexithymic individuals are impaired in their ability to communicate and decode emotions. Neurological imaging has been promising, however. Several previous studies have found differences in activation for alexithymic versus nonalexithymic individuals while viewing images of negative affect (Moriguchi et al., 2007; Walker, O'Connor, & Schaefer, 2011). However, few studies have extended this line of research to include differences of brain activation while viewing positive affect. Thus, this study seeks to begin to correct this oversight by looking for group differences in brain activation between alexithymic and nonalexithymic individuals while viewing images of individuals communicating positive affect. Importantly, if alexithymia represents a deficit in emotion processing, it would be expected to impair neural reactivity to images of positive affect but not to affectively neutral images.

Our mission in this study is threefold. First, we add to the research on the neurological differences between alexithymic and nonalexithymic individuals while viewing images of positive and neutral affect. Second, since alexithymia is highly comorbid with other conditions on the autism spectrum, we hope to translate our findings to those other conditions, thus being able to develop possible interventions helping these groups. Finally, we aim to advance understanding of the physiology of

communication behavior. As others have argued (Cacioppo, Tassinary, & Berntson, 2007), communication is an embodied act, and we believe that it is essential for our field to learn more about how our physiology influences the way that we understand communication phenomena.

To further develop our arguments, we first overview alexithymia, including previous research delving into the neurological components of this condition. We then discuss parts of the brain implicit in the emotional experience, which will lead us to several testable hypotheses.

Alexithymia

Sifneos (1973) conceived of alexithymia (a lack of words for emotions) after noticing a number of individuals who seemed emotionally deficient during therapy sessions. There are four elements of alexithymia, including (a) being unable to understand and process emotion; (b) being unable to communicate emotions to others; (c) processing events and behaviors externally; and (d) engaging in limited amounts of fantasy (Taylor, Bagby, & Parker, 1997). Alexithymic individuals are less able to decode emotions, and thus show less ability to empathize with others (Bird et al., 2010; Moriguchi et al., 2007). Alexithymic individuals also have greater levels of difficulty than nonalexithymics in making lexical decisions in communicating emotions (Suslow & Junghaans, 2002). Alexithymia is commonly defined as a somewhat stable trait, with high levels of alexithymia affecting around 7–10% of the population (Joukamaa et al., 2007; Salminen, Saarijärvi, Toikka, Kauhanen, & Äärelä, 2006). However, it is important to note that scholars usually operationally define alexithymia as a continuous variable, where everyone is more or less alexithymic. Alexithymia is not deemed as a psychiatric disorder in the DSM-IV, although it shows high degrees of comorbidity with other pervasive developmental disorders such as autism spectrum disorders and Asperger's syndrome (Bird et al., 2010; Hill, Berthoz, & Frith, 2004).

A growing body of literature has linked alexithymia to a host of physical, psychological, and relational deficiencies. These have included inverse correlations to indices such as health-related quality of life (Mattila et al., 2009), and positive correlations with depressive disorders (Kugel et al., 2012), chronic pain (Hosoi et al., 2010), cardiovascular reactivity (Lumley, Stettner, & Wehmer, 1996), and risks for addictive behaviors (De Berardis et al., 2009). This line of research has recently been extended to include communicative deficits in affection and relational messages such as intimacy and composure (Hesse & Floyd, 2011, 2008). While these studies support the overall claim that alexithymia is correlated with negative biopsychosocial health, they do not address potential physiological differences between alexithymic and nonalexithymic individuals while experiencing emotions. One of these differences is potentially found in the brain.

Several studies have begun to examine the influence of alexithymia on brain processing with both emotional recognition and regulation. For example, one recent

study found inverse correlations between activation in several areas of the brain (e.g. the insula and amygdala) and alexithymia while individuals viewed masked faces showing emotional expressions (Reker et al., 2010). Another asked individuals to engage in emotion regulation while viewing neutral and negative emotional images, finding an inverse correlation between alexithymia and emotion-related activity during suppression (Walker, O'Connor, & Schaefer, 2011). Other recent studies have found differences in brain activation between alexithymic and nonalexithymic participants while viewing negative words concerning interpersonal relationships (Miyake et al., 2012), inverse correlations between alexithymia and neural response to angry facial displays in the right caudate (Lee et al., 2011), inverse correlation between brain activity and alexithymia while participants are viewing negative images and instructed to "feel what the people in the images are feeling" (Moriguchi et al., 2007), though there appears to be no difference in brain morphology (Heinzel et al., 2012). One recent study examined group differences while viewing both happy and fearful facial expressions, finding a positive correlation between alexithymia and activation in the anterior cingulated cortex while individuals viewed both kinds of emotional expressions (Heinzel et al., 2010).

This study seeks to extend this research by examining group differences between high- and low-alexithymic individuals while viewing images with people showing both neutral and positive emotions. Participants will also be asked to try to feel what the people in the images are feeling. Before explicating our specific hypotheses, we will now review some of the specific areas of the brain that are of concern.

Neurological structures involved in emotion processing

Via the limbic system, several structures in the brain are implicated in the processing of emotional stimuli. Using the language of neuroimaging research, we refer to these as the neurological regions of interest (ROIs). In this section, we identify the ROIs we expect to be activated in response to emotionally expressive faces.

Superior temporal gyrus

The superior temporal gyrus is located in the left and right temporal lobes. Several studies have confirmed its activation in response to facial expressions of emotion (Batty & Taylor, 2003). In an fMRI study with 63 healthy and 32 schizophrenic adults, Radua et al. (2010) examined neural activation in response to individual features of a fearful face—eye behavior, eyebrow behavior, and mouth behavior—that were identified in principal components analyses. The healthy adults showed activation in the left superior temporal gyrus both to eye and mouth behaviors, a response that was not replicated by schizophrenic participants. Similarly, Phillips et al. (1998) demonstrated superior temporal gyrus activation in response to both facial and vocal expressions of disgust. Data from Haxby, Hoffman, and Gobbini (2000) indicate that the superior temporal gyrus is particularly sensitive to dynamic rather than

static facial features, suggesting a more involved role in adjudicating facial emotion than, for instance, facial identity.

Insular cortex

The brain's insular cortex, or *insula*, is a portion of the cerebral cortex that is folded in each hemisphere between the frontal and temporal lobes. The insula regulates multiple body functions, including motor control (Fink, Frackowiak, Pietrzyk, & Passingham, 1997), maintenance of homeostasis (Critchley, 2005), immune regulation (Ramírez-Amaya & Bermúdez-Rattoni, 1999), and self-perception (Tsakiris, Hesse, Boy, Haggard, & Fink, 2007). Stein, Simmons, Feinstein, and Paulus (2007) presented healthy and anxiety-prone college students with angry, fearful, or happy faces and documented increased insular activation in response to the photos for anxious participants. Wicker et al. (2003) found insular activation in response to photos of disgusted faces, and Moriguchi et al. (2007) found that alexithymics had greater right insular activation than nonalexithymics in response to photos of hands and feet in painful situations.

Amygdala

The amygdala is composed of two groups of nuclei—*amygdalae*—located in the median temporal lobes. Considered a principal component of the limbic system, the amygdala is active in the processing and memory of emotional reactions (Amunts et al., 2005). Multiple studies have demonstrated amygdalar activation in response to emotional faces (Jiang & He, 2006). Comparing responses to happy and sad faces among healthy adults, Killgore and Yurgelun-Todd (2004) documented amygdalar reaction to happy faces only. Kugel et al. (2008) found, however, that alexithymic adults responded with amygdalar activation both to happy and to sad faces, and that right amygdalar response to sad faces correlated with their self-reported difficulty at identifying emotions. The amygdala similarly responds to signals of fear (Liddell et al., 2005; Rauch et al., 2007).

Fusiform gyrus

The fusiform gyrus is located in the temporal lobe as part of occipitotemporal Area 37, also known as Brodmann Area 37. Among its primary functions is the recognition of bodies and faces (Kanwisher, McDermott, & Chun, 1997). The cortex around the fusiform gyrus is known as the *fusiform face area* because it is activated approximately twice as strongly by the sight of faces as by the sight of other objects (Grill-Spector, Knouf, & Kanwisher, 2004). Whereas the fusiform gyrus plays a principal role in processing facial identity (Haxby, Hoffman, & Gobbini, 2000), it also responds to facial expressions of emotion when the faces are visible. Jiang and He (2006) found that viewing both fearful and emotionally neutral faces produced activation in the fusiform face area but that viewing scrambled faces did not. Radua et al. (2010) similarly found that fearful and neutral faces increased activation of the right fusiform gyrus in healthy adults, although the same photos decreased activation of the right fusiform gyrus in schizophrenic adults.

Additional ROIs

Additional neurological structures identified as emotion-recognition systems include the orbitofrontal cortex and the superior colliculus (Adolphs, 2002a). The orbitofrontal cortex, a prefrontal cortex region in the brain's frontal lobes, is involved in sensory integration, decision making, and emotion (Kringelbach, 2005). Several experiments have demonstrated activation of the orbitofrontal cortex in response to photos of emotional expression (Vuilleumier, Armony, Driver, & Dolan, 2001), particularly when viewers are asked to identify the emotion being displayed (Narumoto et al., 2000). The superior colliculus, also known as the optic tectum, is a paired structure forming a major component of the midbrain that produces shifts in gaze (Sprague, 1996). Research suggests that the superior colliculus is activated at the onset of an emotional stimulus, such as a photograph of an emotional expression, when perceptual processing begins and before the body's emotional reaction ensues (Adolphs, 2002b).

Synthesis

Alexithymia limits individual ability to decode and communicate emotions. A growing body of literature is pointing to neurological differences between high- and low-alexithymic individuals while viewing emotional images (Moriguchi et al., 2007). This study seeks to extend this research through examining positive and neutral affect. Specifically, we expect different levels of activation between high- and low-alexithymic individuals in the brain regions implicated in emotional processing while the individuals view images of positive affect. However, our goal is to understand the differences due primarily to images of positive affect. To do so, we want to control for any differences found with images of neutral affect. Thus, the question of this study lies in the group difference in activation of positive affect *minus the difference in activation of neutral affect*. This leads to our hypothesis: Controlling for levels of activation due to neutral affect images, alexithymic individuals experience lower levels of activation in our ROIs in the brain (e.g. fusiform gyrus) due to positive affect images than do nonalexithymic individuals.

Methods

Recruitment and prescreening

Participants were recruited from a large university in the Midwestern United States. We advertised the study through a campus-wide e-mail delineating the purpose and expectations of the study. Prospective participants were informed that they would receive $50 for completion of the study, as well as a link to an online questionnaire, posted on the Web site SurveyMonkey.com.

The online questionnaire began with a brief note stating that participants were consenting to their participation in the questionnaire part of the study by filling out and returning the questionnaire. Interested participants then completed the online questionnaire, which primarily consisted of the measurement of trait alexithymia. The

final page of the questionnaire elicited participants' contact information, including their e-mail address and a telephone number. The participants were informed that they would be contacted soon if they qualified to continue with the second part of the study.

Trait alexithymia was measured with the Toronto Alexithymia Scale (TAS-20: Bagby, Parker, & Taylor, 1994), which has previously shown a high level of validity and reliability in several studies (for review, see Taylor & Bagby, 2004). Items on the TAS-20 include, "I am often confused about what emotion I am feeling," and "People tell me to describe my feelings more." Specifically, earlier tests have supported content validity, concurrent validity, and predictive validity by correlating alexithymia with a host of outcome measures (see Bagby, Parker, & Taylor, 1994; Taylor & Bagby, 2004). The TAS-20 has also proven sufficiently reliable, with excellent test–retest findings and high levels of internal consistency (Salminen et al., 2006). Participant reports of alexithymia showed high levels of reliability ($\alpha = .94$).

Inclusion and exclusion criteria

Our main inclusion criteria were based on the participant score on the TAS-20. Earlier studies classified an individual as alexithymic if his or her score was greater than or equaled the 60th percentile score possible on the scale (e.g., a score of 60 out of 100 on a 5-point scale). While this is not a clinical definition, numerous alexithymia papers have used this cutoff in their research, with the number initially proposed by two of the predominant alexithymia scholars, Bagby and Taylor, in their 1997 piece regarding the validation of the alexithymia construct (Joukamaa et al., 2003, 2007). We thus endeavored to create the alexthymic group from individuals who scored near or above a sum of 84 on the trait alexithymia measure since we used a 7-point scale (so 84 out of 140 is the 60th percentile). To ensure that there would be a significant difference on mean alexithymia score between the alexithymic and nonalexithymic groups, individuals were only qualified as nonalexithymic if they scored near or below a sum of 50 on the trait alexithymia scale (right around the 35% possible score on the TAS-20). This cut-off point was used in one previous study that did find several group differences between alexithymic and nonalexithymic participants (Hesse & Floyd, 2011).

Prospective participants were excluded from participating if they reported ever having been diagnosed with clinical depression, anxiety or mood disorders, or personality disorders, as those could be possible confounds with the effect of alexithymia on the interaction. Other exclusions included individuals with metal fillings and claustrophobia, since they would be unable to go inside the MRI machine.

Participants

A large number of individuals filled out the online questionnaire ($N = 302$), consisting of 227 women and 68 men (7 individuals declined to give their sex). Out of that sample, two research assistants first screened each questionnaire to ensure that the prospective participant had completed all of the necessary information, including

the alexithymia measure, the contact information, and the exclusion criteria. They then computed the sum of the trait alexithymia measure for each prospective participant. A small portion qualified as alexithymic (a sum score of 80 or higher on the trait alexithymia scale), with 3% qualifying for the male/alexithymic group and another 9% qualifying for the female/alexithymic group. Another portion qualified as nonalexithymic (a sum score of 50 or below on the trait alexithymia scale), with 27% qualifying on the male/nonalexithymic group and 10% qualifying for the female/nonalexithymic group.

We contacted the prospective participants, beginning with the individuals who scored the highest and lowest, respectively, on the TAS-20, to invite their participation in the laboratory study. If they agreed, they were scheduled for a future laboratory session. Twenty individuals participated in the study. Those individuals were placed in one of four groups of five participants: male/alexithymic ($M = 85.00$, $SD = 5.66$), male/nonalexithymic ($M = 38.20$, $SD = 5.02$), female/alexithymic ($M = 95.40$, $SD = 11.57$), and female/nonalexithymic ($M = 38.00$, $SD = 5.00$). The sample was relatively young ($M = 22.55$, $SD = 6.68$), ranging from 18 to 46 years old. The racial composition of this sample was 90% Caucasian and 10% Latino. The alexithymic group scored significantly higher on the TAS-20 than the nonalexithymic group, $t(18) = 14.67$, $p < .001$.

Procedure

The following procedure was a subset of a larger study, and so only those parts of the study relevant to the current analyses will be discussed. Participants took part in one fMRI session, which consisted of 6 series of 20 blocks of images. Participants were exposed to three types of images. The two types relevant to this study were neutral affect and positive affect. Neutral affect images were composed of individuals showing no facial expression of emotion, while positive affect images consisted of individuals showing positive expressions of emotion, including smiling and laughing. Some of the images for both conditions came from the International Affective Picture System (IAPS: Lang, Bradley, & Cuthbert, 2008). However, the majority of the images were procured by pictures taken by one of the research assistants of individuals showing neutral or positive affect.[1]

Since the majority of the images had been procured for this specific study, the images were pilot-tested by a separate sample of 237 participants to check for differences in affect and arousal, since previous scholars have stated that conditions that raise positive affect also likely increase arousal (Ashby, Valentin, & Turken, 2002). As expected, the positive affect condition ($M = 3.66$, $SD = 1.32$) was significantly higher on positive affect than the neutral condition ($M = 1.97$, $SD = 0.74$), $t(236) = 24.16$, $p < .001$. The positive affect condition ($M = 2.16$, $SD = 1.09$) was also significantly higher on arousal than the neutral condition ($M = 1.61$, $SD = 0.64$), $t(236) = 10.42$, $p < .001$.

The participants were instructed before the beginning of the session to attempt to feel what the individuals in the images were feeling. The order of conditions

and images were randomized within the session, with no image being presented more than once through the experiment. After the imaging runs were completed, participants were asked to go to a separate computer and look at each image again, scoring the image for levels of affect and arousal. Participants were then debriefed, paid, and sent on their way. Each complete session took roughly 60 minutes.

Manipulation check

Two manipulation checks were needed for this study. First, the participants rated each image on the level of affect. As predicted, participants said that the neutral affect images ($M = 2.21$, $SD = 0.66$) showed significantly less affect than the positive affect images ($M = 4.51$, $SD = 0.88$), $t(19) = -12.36$, $p < .001$. Second, participants rated each image on level of arousal. As predicted, participants said that the neutral affect images ($M = 1.52$, $SD = 0.47$) showed significantly less arousal than the positive affect images ($M = 1.84$, $SD = 0.80$), $t(19) = -2.76$, $p = .01$.

Data acquisition and analyses

Magnetic resonance imaging data were acquired on a 3 T Siemens Trio MRI scanner (Erlangen, Germany). Anatomical T1-weighted MPRAGE images were acquired in the sagittal plane with the following parameters: TR = 1,920 milliseconds, TE = 2.92 milliseconds, TI = 1,000 milliseconds, flip angle = 9°, in plane voxel size = 1 mm × 1 mm, slice thickness = 1 mm and 0.5 mm gap, slice number = 176, and FOV = 256 mm × 256 mm. Functional BOLD T2*-weighted EPI images were acquired in the same session parallel to the AC–PC plane with the following parameters: TR = 2,000 milliseconds, TE = 30 milliseconds, flip angle = 90°, in plane voxel size = 4 mm × 4 mm, slice thickness = 4 mm with no gap, slice number = 32, and FOV = 256 mm × 256 mm. A total of six functional runs were acquired for every subject and each run consisted of a total of 152 volume images. No data were collected in the first two volumes and each run began with a fixation period allowing for T1 effects to stabilize before acquisition of stimulus-related data. Images were presented in a block design grouped on emotional salience and separated by 16,000 milliseconds fixation rest periods. Two blocks of each image type, positive, intimate, and neutral, were presented within each run and were counterbalanced for presentation order. Each block contained eight randomly assigned images presented for 3,500 milliseconds each with a 500 milliseconds prestimulus fixation display. Stimuli were presented on a translucent screen mounted at the rear of the scanner and viewed through a mirror attached to the head coil.

Image processing for this publication compared positive and neutral image blocks and was carried out using FMRIB's Software Library, FSL http://www.fmrib.ox.ac.uk/fsl (Smith et al., 2004; Woolrich et al., 2009). Prestatistic processing consisted of motion correction using MCFLIRT (Jenkinson et al., 2002), nonbrain removal using BET (Smith, 2002), spatial smoothing using a Gaussian

kernel of FWHM 5 mm, and highpass temporal filtering of 100 seconds. Time-series modeling for statistical analysis was carried out using FILM with local autocorrelation correction (Woolrich et al., 2001) and convolved with a double-gamma basis function. All functional data were registered first to each subject's high-resolution T1-weighted anatomical images and then a standard Montreal Neurological Institute, MNI_152, atlas with FLIRT (Jenkinson et al., 2002; Jenkinson & Smith, 2001).

For each subject on each individual run, activation maps were generated for positive, neutral, and the difference between image presentations for the fMRI data by modeling at the first level with explanatory variables (EVs) for each image type as well as image type comparisons. Activation maps were created by generating z-scores of EV model fit to hemodynamic response data from each voxel within the brain field of view. Temporal filtering was applied and the resulting first-level contrasts of parameter estimates (COPEs), corresponding to individual analyses of each functional run, were input to higher-level analysis with fixed effects for within-subjects averaging of runs at the second level. Averaging at the second level was completed in order to generate more robust representations of the hemodynamic response under each stimulus condition. Second-level COPEs were input to higher-level analysis with mixed effects for between-subjects averaging at the third level using FLAME 1 (Beckmann et al., 2003; Woolrich et al., 2004). Third-level analyses generated group-based, alexithymic versus nonalexythymic, activation maps and allowed variability associated with age, gender, and affection to be accounted for within the model. All levels were thresholded for clusters at $Z > 2.3$, $p < .05$ (Worsley, 2001).

Results

Our hypothesis predicted a group difference between our alexithymic ($n = 10$) and our nonalexithymic ($n = 10$) conditions in levels of brain activation while viewing images of positive affect after controlling for differences of neutral affect. Table 1 shows any significant findings ($p < .05$) in terms of activation due to positive affect controlling for neutral affect for the alexithymic group, the nonalexithymic group, and the group difference tests. We found a host of differences where the nonalexithymic group showed significantly greater levels of activation than the alexithymic group. Specifically, we found lower hemodynamic activity in the alexithymic versus nonalexithymic condition in the left inferior temporal/temporal occipital fusiform, the right lingual/parahippocampal gyrus, the left temporal occipital inferior temporal gyrus, the left lateral occipital fusiform gyrus, the left cerebellum anterior lobe, the left lateral temporal occipital fusiform gyrus, the left superior temporal gyrus, the left middle temporal gyrus, the left amygdala/planum polare, the left hippocampus, the left insular cortex, the right hippocampus, the right amygdala, and the right subcallosal gyrus. Pictoral examples of these differences in activation are found in Figures 1 and 2. As we did not find differences in every area of the brain associated with emotional processing, H1 is partially supported.

Table 1 Significant Coordinates, Z Scores, and Effect Sizes for Brain Areas Activated in Response to Positive Affect Images Controlling for Neutral Affect After Regressing out Trait Affection, Age, and Gender

Anatomical Region	Peak Activation Coordinates (MNI)			Max z Score	*t* Value	Cohen's *d*	Effect Size, r	Number Voxels of Above Threshold
	X	*y*	*z*					
Alexithymic								1,870
R Middle Occipital Gyrus/Occipital Pole	24	−98	12	4.29	6.59	3.66	0.88	
R Cuneus/Occipital Pole	14	−104	8	4.01	5.82	3.23	0.85	
R Occipital Fusiform Gyrus	38	−68	−14	3.85	5.42	3.01	0.83	
Nonalexithymic								27,772
R Cuneus/Occipital Pole	26	−94	8	4.74	8.05	4.47	0.91	
L Temporal Occipital Fusiform Gyrus	−42	−56	−12	4.56	7.4	4.1	0.9	
L Lingual Gyrus/Occipital Pole	−2	−102	−2	4.55	7.39	4.1	0.9	
L Hippocampus	−24	−14	−20	4.51	7.24	4.01	0.9	
L Medial Frontal Gyrus	−2	52	−10	3.57	4.79	2.66	0.8	
R Superior Frontal Gyrus/Frontal Pole	4	58	22	3.49	4.62	2.56	0.79	
L Paracingulate/Medial Frontal Gyrus	−4	56	10	3.24	4.13	2.29	0.75	
R Medial Frontal Gyrus/Frontal Pole	6	58	0	3.18	4.02	2.23	0.74	
L Anterior Cingulate/Paracingulate Gyrus	−6	38	−10	3.14	3.94	2.19	0.73	
Alexithymic > Nonalexithymic								0
N/A								
Nonalexithymic > Alexithymic								6,340
L Temporal Occipital Fusiform Gyrus	−42	−56	−12	4.08	5.99	3.32	0.86	
R Lingual/Parahippocampal Gyrus	18	−40	−4	3.91	5.57	3.09	0.84	
L Temporooccipital Inferior Temporal Gyrus	−40	−52	−8	3.81	5.33	2.96	0.83	

Table 1 Continued

Anatomical Region	Peak Activation Coordinates (MNI)			Max z Score	*t* Value	Cohen's *d*	Effect Size, r	Number Voxels of Above Threshold
	X	*y*	*z*					
L Lateral Occipital Fusiform Gyrus	−52	−66	−14	3.63	4.91	2.72	0.81	
L Cerebellum Anterior Lobe	−24	−44	−26	3.52	4.68	2.6	0.79	
L Lateral Temporooccipital Fusiform Gyrus	−46	−68	−12	3.48	4.6	2.55	0.79	
L Superior Temporal Gyrus/Temporal Pole	−34	2	−24	3.56	4.77	2.65	0.8	
L Temporooccipital Middle Temporal Gyrus	−38	−46	6	3.52	4.68	2.6	0.79	
L Amygdala/Planum Polare	38	−6	−22	3.36	4.35	2.41	0.77	
L Hippocampus	−24	−14	−20	3.32	4.27	2.37	0.76	
L Insular Cortex/Inferior Frontal Gyrus	−34	8	−18	3.24	4.12	2.29	0.75	
L Insular Cortex/Claustrum	−38	−18	−2	3.18	4.01	2.22	0.74	
R Hippocampus	26	−12	−22	3.81	5.33	2.96	0.83	
R Amygdala	30	2	−22	3.21	4.07	2.26	0.75	
R Subcallosal Gyrus/Frontal Orbital Cortex	28	8	−16	2.94	3.6	2	0.71	

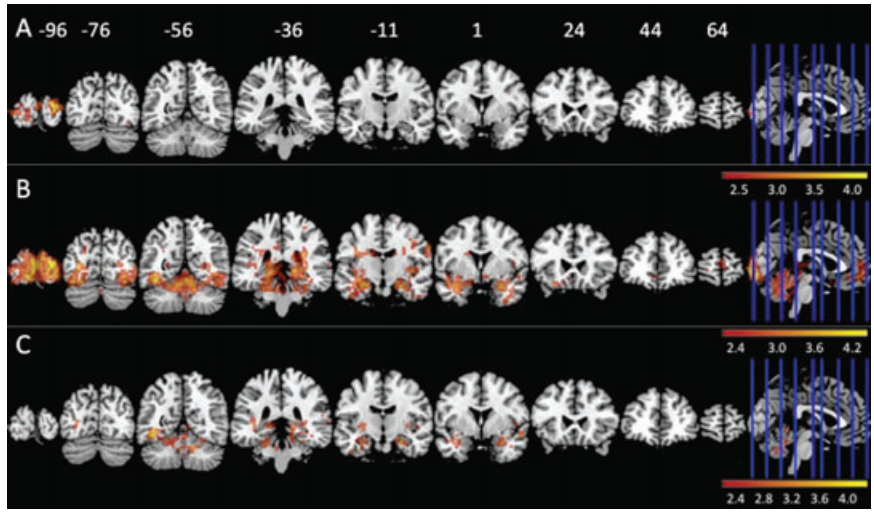

Figure 1 Coronal view of brain activation while viewing positive affect images controlling for neutral images, (a) alexithymic, (b) nonalexithymic, and (c) nonalexithymic > alexithymic.

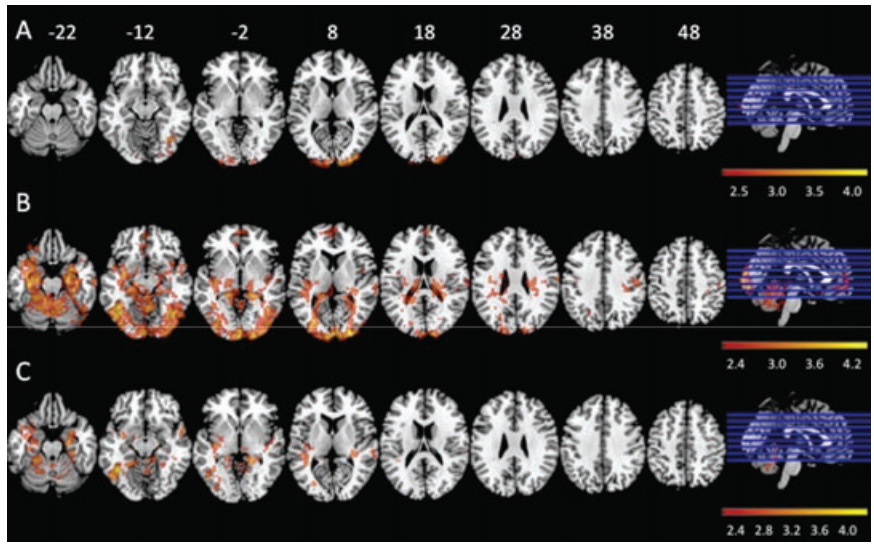

Figure 2 Axial view of activation while viewing positive affect images controlling for neutral images, (a) alexithymic, (b) nonalexithymic, and (c) nonalexithymic > alexithymic.

Discussion

The ability to decode emotional displays accurately—an evolutionarily adaptive ability—is impaired by the psychological trait alexithymia. The ability of any organism to interpret negative emotion displays accurately—and to initiate an

appropriate peripheral nervous system response—is unquestionably advantageous with respect to survival. For the sake of procreation, cooperation, and other social benefits, however, the ability to interpret and respond appropriately to positive emotion displays is equally advantageous. A good deal of research has demonstrated that alexithymic individuals respond with weaker patterns of neural activation to signals of negative emotion, such as fear and disgust, than do nonalexithymics. This study sought to determine whether alexithymic individuals face similar detriments, relative to their nonalexithymic counterparts, in their neural reaction to scenes of positive emotion.

The results document a pattern of weaker activation in response to scenes of positive emotion for alexithymics, relative to nonalexithymics, in several neural structures relevant to the perception and processing of social and emotional signals. Some such structures—including the superior temporal gyrus, insular cortex, amygdala, and fusiform gyrus—were hypothesized to see weaker activation in alexithymics on the basis of extant research relevant to their function. Other such structures, such as the inferior temporal gyrus, lingual gyrus, and parahippocampal gyrus, play active roles in processing visual cues—such as the nonverbal emotion displays in the photographs—and in retrieving stored memory—such as that required to connect visual cues with meaning.

Notably, the pattern of weaker neural activation for alexithymics did not extend to scenes that were emotionally neutral. Considered in concert with the results of Wicker et al. (2003), Phillips et al. (1998), and others relative to negative emotion, these findings suggest that the alexithymic deficit is not undifferentiated, but is specific to social cues with an identifiable emotional valence. This growing body of research leads to an interesting conclusion regarding the conceptualization of the TAS-20. As currently constructed, the scale asks participants questions regarding difficulty identifying their own feelings. However, the scale includes no items regarding an individual deficit in identifying emotion-laden social cues. Future alexithymia research might want to revise the scale to include the apparent social deficit inherent in alexithymia.

Implications of findings

As stated earlier, communication scholars have built an impressive literature regarding the importance of accurately decoding emotions to biopsychosocial health. Recent articles have focused on negative communicative correlates with the psychological construct of alexithymia. This study potentially helps our understanding of *why* some individuals have a lesser ability to decode emotions. This knowledge could presumably lead to the possibility of communication-based interventions designed to help neurologically impaired individuals. Basically, the question will be whether communication scholars can help individuals overcome a physiological impairment through learned behavior. Other complementary medicine techniques (though somewhat controversial) have been found to lower levels of alexithymia, including hypnosis (Gay, Hanin, & Luminet, 2008) and group psychotherapy (Levant, Halter, Hayden, & Williams, 2009).

This study complements recent efforts to document the neurological substrates of communication behavior via neuroimaging technologies such as fMRI (Brefcyznski-Lewis, 2011) and electroencephalography (Lewis, Heisel, Reinhart, & Tian, 2011; Pence, Heisel, Reinhart, Tian, & Beatty, 2011). Whereas many methods can identify the behavioral and perceptual differences between, for instance, those high and low in alexithymia, neuroimaging techniques contribute to an understanding of how such differences manifest themselves at a physiological level. Together with the growing literature on communication correlates in other physiological domains, such as the cardiovascular, endocrine, and immune systems (for recent reviews, see Boren & Veksler, 2011; Floyd & Afifi, 2012), this research illuminates social and communicative behavior as interacting in a sophisticated manner with the biological self.

Efforts to understand that interaction can uncover new ground in our understanding not only of emotion-processing deficits such as alexithymia, but also of affective communication, nonverbal signaling, health communication, and interpersonal behavior in general. As Floyd and Afifi (2012) intimated, it is both unnecessary and inaccurate to conceptualize communication as a disembodied entity, affecting and being affected by social forces alone. As studies of the present sort illustrate, communication behaviors also interact with the body at a fundamentally physical level. Identifying and understanding those interactions, as this study attempts to do, is a necessary first step toward treating conditions such as alexithymia more successfully and also toward shaping messages that are responsive to individual differences in message-processing ability.

Conclusions

While this study possessed several strengths, it is necessary to point out weaknesses that potentially mitigate conclusions that could be reached from our data. The sample was relatively homogenous for age and ethnicity, and was very healthy. Future studies might want to examine these differences using clinical samples, especially since alexithymia is related to substance abuse and psychological illnesses (De Berardis et al., 2009). It was also a small sample size, even for imaging research, which limits the generalizability of the study. However, several previous studies on imaging and alexithymia have used similar sample sizes (Decety, Michalska, Akitsuki and Lahey, 2009; Moll et al., 2005). Future studies should attempt to see if the current findings are replicable with larger sample sizes. Future research should also examine the impact of such variables as empathy and perspective taking on these findings. Using brain-imaging procedures constrained us in terms of the type of communication under review. It is very difficult, for example, to assess momentary brain activation while viewing verbal emotional communication. The participants were not able to engage in any face-to-face communication with individuals while being scanned, limiting our ability to understand what is occurring at the neurological level while an individual is actually engaging in communication. Finally, it is proper to pose any alternate explanations that could potentially explain our findings. One

possibility would be that alexithymic participants are impaired in general in terms of information processing, without paying attention to emotional salience. This could be due to less attention paid to the stimuli as reflected in the frontal activation that was found coupled with decreased consolidation of the information as seen in the hippocampal activation. Alexithymic participants could also be worse at processing faces or interpersonal scenes as seen through the decreased fusiform activation coupled with the frontal/hippocampus activation.

Even with these limitations, we are excited about the outcome of this study, as it allows us an initial look at how communication scholars can examine phenomena through functional imaging. These findings serve as evidence toward a physiological explanation for individual differences in understanding emotional communication. This could lead to several fruitful lines of research for examining other areas where questions of emotion and communication intersect, including communication apprehension, uncertainty management, or conflict.

Note

1 Our goal with the images was to show people engaging in affective communication in a real-world setting. As such, the images varied in respect to the directness of gaze, the orientation of the image, and the number of people in each image. We did not control for the racial identity or sex of the individuals in the photographs.

References

Adolphs, R. (2002a). Neural systems for recognizing emotion. *Current Opinion in Neurobiology*, **12**, 169–177. doi: 10.1016/S0959-4388(02)00301-X

Adolphs, R. (2002b). Recognizing emotion from facial expressions: Psychological and neurological mechanisms. *Behavioral and Cognitive Neuroscience Reviews*, **1**, 21–61. doi: 10.1177/1534582302001001003

Amunts, K., Kedo, O., Kindler, M., Pieperhoff, P., Mohlberg, H., Shah, N., et al. (2005). Cytoarchitectonic mapping of the human amygdala, hippocampal region and entorhinal cortex: Intersubject variability and probability maps. *Anatomy and Embryology*, **210**(5–6), 343–352. doi: 10.1007/s00429-005-0025-5

Ashby, F. G., Valentin, V. V., & Turken, A. U. (2002). The effects of positive affect and arousal on working memory and executive attention: Neurobiology and computational models. In S. Moore & M. Oaksford (Eds.), *Emotional cognition: From brain to behavior* (pp. 245–288). Philadelphia, PA: John Benjamins Publishing Co.

Bagby, R. M., Parker, J. D. A., & Taylor, G. J. (1994). The twenty-item Toronto Alexithymic Scale—I. Item selection and cross-validation of the factor structure. *Journal of Psychosomatic Research*, **38**, 23–32. doi: 10.1016/0022-3999(94)90005-1

Bagby, R. M., & Taylor, G. (1997). Construct validation. In G. J. Taylor, R. M. Bagby, & J. D. A. Parker (Eds.), *Disorders of affect regulation: Alexithymia in medical and psychiatric illness* (pp. 46–66). Cambridge, England: Cambridge University Press.

Batty, M., & Taylor, M. J. (2003). Early processing of the six basic facial emotion expressions. *Cognitive Brain Research*, **17**, 613–620. doi: 10.1016/S0926-6410(03)00174-5

Beckmann, C. F., Jenkinson, M., & Smith, S. M. (2003). General multilevel linear modeling for group analysis in FMRI. *NeuroImage*, **20**, 1052–1063. doi: 10.1016/S1053-8119(03)00435-X

Bird, G., Silani, G., Brindley, R., White, S., Frith, U., & Singer, T. (2010). Empathic brain responses in insula are modulated by levels of alexithymia but not autism. *Brain*, **133**, 1515–1525. doi: 10.1093/brain/awq060

Boren, J. P., & Veksler, A. E. (2011). A decade of research exploring biology and communication: The brain, nervous, endocrine, cardiovascular, and immune systems. *Communication Research Trends*, **30**, 3–24.

Brefcyznski-Lewis, J. A. (2011). Neuroimaging: Directions and potentials for communication research. *Communication Research Reports*, **28**, 196–204. doi: 10.1080/08824096.2011.566106

Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (2007). Psychophysiological science: Interdisciplinary approaches to classic questions about the mind. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 1–18). New York, NY: Cambridge University Press.

Carton, J. S., Kessler, E. A., & Pape, C. L. (1999). Nonverbal decoding skills and relationship well-being in adults. *Journal of Nonverbal Behavior*, **23**, 91–100. doi: 10.1023/A:1021339410262

Critchley, H. D. (2005). Neural mechanisms of autonomic, affective, and cognitive integration. *Journal of Comparative Neurology*, **493**, 154–166. doi: 10.1002/cne.20749

Darwin, C. (1859). *On the origin of species by means of natural selection*. New York, NY: Philosophical Library.

Darwin, C. (1872). *The expression of emotions in man and animals*. New York, NY: Philosophical Library.

De Berardis, D., D'Albenzio, A., Gambi, F., Sepede, G., Valchera, A., Conti, C. M., et al. (2009). Alexithymia and its relationships with dissociative experiences and internet addiction in a nonclinical sample. *CyberPsychology & Behavior*, **12**, 67–69. doi: 10.1089/cpb.2008.0108

Decety, J., Michalska, K. J., Akitsuki, Y., & Lahey, B. B. (2009). Atypical empathic responses in adolescents with aggressive conduct disorder: A functional MRI investigation. *Biological Psychology*, **80**, 203–211. doi: 10.1016/j.biopsycho.2008.09.004

DiMatteo, M. R., Hays, R. D., & Prince, L. M. (1999). Relationship of physicians' nonverbal communication skill to patient satisfaction, appointment noncompliance, and physician workload. *Health Psychology*, **5**, 581–594. doi: 10.1037/0278-6133.5.6.581

Egolf, D. B. (2012). *Human communication and the brain: Building the foundation for the field of neurocommunication*. Lanham, MD: Lexington Books.

Ekman, P. (2003). Darwin, deception, and facial expression. *Annals of the New York Academy of Sciences*, **1000**, 205–221. doi: 10.1196/annals.1280.010

Fink, G. R., Frackowiak, R. S., Pietrzyk, U., & Passingham, R. E. (1997). Multiple nonprimary motor areas in the human cortex. *Journal of Neurophysiology*, **77**, 2164–2174.

Floyd, K. (2006). *Communicating affection: Interpersonal behavior and social context*. Cambridge, England: Cambridge University Press.

Floyd, K., & Afifi, T. D. (2012). Biological and physiological perspectives on interpersonal communication. In M. L. Knapp & J. A. Daly (Eds.), *The handbook of interpersonal communication* (4th ed., pp. 87–127). Thousand Oaks, CA: Sage.

Gay, M. C., Hanin, D., & Luminet, O. (2008). Effectiveness of an hypnotic imagery intervention on reducing alexithymia. *Contemporary Hypnosis*, **25**, 1–13. doi: 10.1002/ch.344

Giles, H. (2008). Communication accommodation theory. In D. O. Braithwaite & L. A. Baxter (Eds.), *Engaging theories in interpersonal communication: Multiple perspectives*. Thousand Oaks, CA: Sage.

Grill-Spector, K., Knouf, N., & Kanwisher, N. (2004). The fusiform face area subserves face perception, not generic within-category identification. *Nature Neuroscience*, **7**, 555–562. doi: 10.1038/nn1224

Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in Cognitive Science*, **4**, 223–233.

Heinzel, A., Minnerop, M., Schäfer, R., Müller, H. W., Franz, M., & Hautzel, H. (2012). Alexithymia in healthy young men: A voxel-based morphometric study. *Journal of Affect Disorders*, **136**, 1252–1256.

Heinzel, A., Schäfer, R., Müller, H. W., Schieffer, A., Ingenhag, A., Eickhoff, S. B., et al. (2010). Increased activation of the supragenual anterior cingulated cortex during visual emotional processing in male subjects with high degrees of alexithymia: An event-related fMRI study. *Psychotherapy and Psychosomatics*, **79**, 363–370.

Hesse, C., & Floyd, K. (2008). Affectionate experience mediates the effects of alexithymia on mental health and interpersonal relationships. *Journal of Social and Personal Relationships*, **5**, 793–810. doi: 10.1177/0265407508096696

Hesse, C., & Floyd, K. (2011). The impact of alexithymia on initial interactions. *Personal Relationships*, **18**, 453–470. doi: 10.1111/j.1475.6811.2010.01311.x

Hill, E., Berthoz, S., & Frith, U. (2004). Brief report: Cognitive processing of own emotions in individuals with autistic spectrum disorder and in their relatives. *Journal of Autism and Developmental Disorders*, **34**, 229–235.

Hosoi, M., Molton, I. R., Jensen, M. P., Ehde, D. M., Amtmann, S., O' Brien, S., et al. (2010). Relationships among alexithymia and pain intensity, pain interference, and vitality in persons with neuromuscular disease: Considering the effect of negative affectivity. *Pain*, **149**, 273–277. doi: 10.1016/j.pain.2010.02.012

Jenkinson, M., Bannister, P., Brady, M., & Smith, S. (2002). Improved optimization for the robust and accurate linear registration and motion correction of brain images. *NeuroImage*, **17**, 825–841. doi: 10.1006/nimg.2002.1132

Jenkinson, M., & Smith, S. (2001). A global optimisation method for robust affine registration of brain images. *Medical Image Analysis*, **5**, 143–156. doi: 10.1016/S1361-8415(01)00036-6

Jiang, Y., & He, S. (2006). Cortical responses to invisible faces: Dissociating subsystems for facial-information processing. *Current Biology*, **16**, 2023–2029. doi: 10.1016/j.cub.2006.08.084

Joukamaa, M., Kokkonen, P., Veijola, J., Läksy, K., Karvonen, J. T., Jokelainen, J., et al. (2003). Social situation of expectant mothers and alexithymia 31 years later in their offspring: A prospective study. *Psychosomatic Medicine*, **65**, 307–312.

Joukamaa, M., Taanila, A., Miettunen, J., Karvonenm, J. T., Koskinen, M., & Veijola, J. (2007). Epidemiology of alexithymia among adolescents. *Journal of Psychosomatic Research*, **63**, 373–376. doi: 10.1016/j.jpsychores.2007.01.018

Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: A module in human extrastriate cortex specialized for face perception. *Journal of Neuroscience*, **17**, 4302–4311.

Keeley-Dyreson, M., Bailey, W., & Burgoon, J. K. (1991). The effects of stress and gender on nonverbal decoding accuracy in kinesic and vocalic channels. *Human Communication Research*, **17**, 584–605. doi: 10.1111/j.1458-2958.1991.tb00245

Killgore, W. D. S., & Yurgelun-Todd, D. A. (2004). Activation of the amygdala and anterior cingulate during nonconscious processing of sad versus happy faces. *NeuroImage*, **21**, 1215–1223. doi: 10.1016/j.neuroimage.2003.12.033

Kringelbach, M. L. (2005). The orbitofrontal cortex: Linking reward to hedonic experience. *Nature Reviews Neuroscience*, **6**, 691–702. doi: 10.1038/nrn1747

Kugel, H., Eichmann, M., Dannlowski, U., Ohrmann, P., Bauer, J., Arolt, V., et al. (2008). Alexithymic features and automatic amygdala reactivity to facial emotion. *Neuroscience Letters*, **435**, 40–44. doi: 10.1016/j.neulet.2008.02.005

Kugel, H., Eichmann, M., Dannlowski, U., Ohrmann, P., Bauer, J., Leweke, F., et al. (2012). Is alexithymia associated with specific mental disorders? *Psychopathology*, **45**, 22–28. doi: 10.1159/000325170

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). *International affective picture system (IAPS): Affective ratings of pictures and instruction manual*. Technical Report A-8, University of Florida, Gainesville, FL.

Lee, B. T., Lee, H. Y., Park, S. A., Lim, J. Y., Tae, W. S., Lee, M. S., et al. (2011). Neural substrates of affective face recognition in alexithymia: A functional magnetic resonance imaging study. *Neuropsychobiology*, **63**, 119–124.

Levant, R. F., Halter, M. J., Hayden, E. W., & Williams, C. M. (2009). The efficacy of alexithymia reduction treatment: A pilot study. *The Journal of Men's Studies*, **17**, 75–84. doi: 10.3149/jms.1701.75

Lewis, R. J., Heisel, A. D., Reinhart, A. M., & Tian, Y. (2011). Trait affection and asymmetry in the anterior brain. *Communication Research Reports*, **28**, 347–355. doi: 10.1080/08824096.2011.615960

Liddell, B. J., Brown, K. J., Kemp, A. H., Barton, M. J., Das, P., Peduto, A., et al. (2005). A direct brainstem-amygdala-cortical 'alarm' system for subliminal signals of fear. *NeuroImage*, **24**, 235–243. doi: 10.1016/j.neuroimage.2004.08.016

Lumley, M. A., Stettner, L., & Wehmer, F. (1996). How are alexithymia and physical illness linked? A review and critique of pathways. *Journal of Psychosomatic Research*, **41**, 505–518. doi: 10.1016/S0022-3999(96)00222-X

Maatila, A. K., Saarni, S. I., Salminen, J. K., Huhtala, H., Sintonen, H., & Joukamaa, M. (2009). Alexithymia and health-related quality of life in a general population. *Psychosomatics*, **50**, 59–68. doi: 10.1176/appi.psy.50.1.59

Miyake, Y., Okamoto, Y., Onoda, K., Shirao, N., Okamoto, Y., & Yamawaki, S. (2012). Brain activation during the perception of stressful word stimuli concerning interpersonal relationships in anorexia nervosa patients with high degrees of alexithymia in an fMRI paradigm. *Psychiatry Research: Neuroimaging*, **201**, 113–119.

Moll, J., de Oliveira-Souza, R., Moll, F. T., Ignácio, F. A., Bramati, I. E., Caparelli-Dáquer, E. M., et al. (2005). The moral affiliations of disgust: A functional MRI study. *Cognitive and Behavioral Neurology*, **18**, 68–78.

Moriguchi, Y., Decety, J., Ohnishi, T., Maeda, M., Mori, T., Nemoto, K., et al. (2007). Empathy and judging other's pain: An fMRI study of alexithymia. *Cerebral Cortex*, **17**, 2223–2234. doi: 10.1093/cercor/bhl130

Narumoto, J., Yamada, H., Iidaka, T., Sadato, N., Fukui, K., Itoh, H., et al. (2000). Brain regions involved in verbal or non-verbal aspects of facial emotion recognition. *NeuroReport*, **11**, 2571–2576. doi: 10.1097/00001756-200008030-00044

Pence, M. E., Heisel, A. D., Reinhart, A., Tian, Y., & Beatty, M. J. (2011). Resting prefrontal cortex asymmetry and communication apprehension, verbal aggression, and other social interaction constructs: A meta-analytic review. *Communication Research Reports*, **28**, 287–295. doi: 10.1080/08824096.2011.615959

Phillips, M. L., Young, A. W., Scott, S. K., Calder, A. J., Andrew, C., Giampietro, V., et al. (1998). Neural responses to facial and vocal expressions of fear and disgust. *Proceedings of the Royal Society of London, B*, **265**, 1809–1817. doi: 10.1098/rspb.1998.0506

Radua, J., Phillips, M. L., Tussell, T., Lawrence, N., Marshall, N., Kalidindi, S., et al. (2010). Neural response to specific components of fearful faces in healthy and schizophrenic adults. *NeuroImage*, **49**, 939–946. doi: 10.1016/j.neuroimage.2009.08.030

Ramírez-Amaya, V., & Bermúdez-Rattoni, F. (1999). Conditioned enhancement of antibody production is disrupted by insular cortex and amygdala but not hippocampal lesions. *Brain, Behavior, and Immunity*, **13**, 46–60. doi: 10.1006/brbi.1998.0547

Rauch, A. V., Ohrmann, P., Bauer, J., Kugel, H., Engelien, A., Arolt, V., et al. (2007). Cognitive coping style modulates neural responses to emotional faces in healthy humans: A 3-T fMRI study. *Cerebral Cortex*, **17**, 2526–2535. doi: 10.1093/cercor/bhl158

Reker, M., Ohrmann, P., Rauch, A. V., Kugel, H., Bauer, J., Dannlowski, U., et al. (2010). Individual differences in alexithymia and brain response to masked emotion faces. *Cortex*, **46**, 658–667. doi: 10.1016/j.cortex.2009.05.008

Salminen, J. K., Saarijärvi, S., Toikka, T., Kauhanen, J., & Äärelä, A. (2006). Alexithymia behaves as a personality trait over a 5-year period in Finnish general population. *Journal of Psychosomatic Research*, **61**, 275–278. doi: 10.1016/j.jpsychores.2006.01.014

Sifneos, P. E. (1973). The prevalence of 'alexithymic' characteristics in psychosomatic patients. *Psychotherapy and Psychosomatics*, **22**, 255–262.

Smith, S. M. (2002). Fast robust automated brain extraction. *Human Brain Mapping*, **17**, 143–155. doi: 10.1002/hbm.10062

Smith, S. M., Jenkinson, M., Woolrich, M. W., Beckmann, C. F., Behrens, T. E. J., Johansen-Berg, H., et al. (2004). Advances in functional and structural MR image analysis and implementation as FSL. *NeuroImage*, **23**, 208–219. doi: 10.1016/j.neuroimage.2004.07.051

Sprague, J. M. (1996). Neural mechanisms of visual orienting responses. *Progress in Brain Research*, **112**, 1–15. doi: 10.1016/S0079-6123(08)63317-8

Stein, M. B., Simmons, A. N., Feinstein, J. S., & Paulus, M. P. (2007). Increased amygdala and insula activation during emotion processing in anxiety-prone subjects. *The American Journal of Psychiatry*, **164**, 318–327. doi: 10.1176/appi.ajp.164.2.318

Suslow, T., & Junghaans, K. (2002). Impairments of emotion situation priming in alexithymia. *Personality and Individual Differences*, **32**, 541–550. doi: 10.1016/S0191-8869(01)00056-3

Taylor, G. J., & Bagby, R. M. (2004). New trends in alexithymia research. *Psychotherapy and Pychosomatics*, **73**, 68–77. doi: 10.1159/000075537

Taylor, G. J., Bagby, R. M., & Parker, J. D. A. (1997). *Disorders of affect regulation: Alexithymia in medical and psychiatric illness*. New York, NY: Cambridge University Press.

Tsakiris, M., Hesse, M. D., Boy, C., Haggard, P., & Fink, G. R. (2007). Neural signatures of body ownership: A sensory network for bodily self-consciousness. *Cerebral Cortex*, **17**, 2235–2244. doi: 10.1093/cercor/bhl131

Vuilleumier, P., Armony, J. L., Driver, J., & Dolan, R. J. (2001). Effects of attention and emotion on face processing in the human brain: An event-related fMRI study. *Neuron*, **30**, 829–841. doi: 10.1016/S0896-6273(01)00328-2

Walker, S., O'Connor, D. B., & Schaefer, A. (2011). Brain potentials to emotional pictures are modulated by alexithymia during emotion regulation. *Cognitive, Affective, and Behavioral Neuroscience*, **11**, 463–475. doi: 10.3758/s13415-011-0042-1

Wicker, B., Keysers, C., Plailly, J., Royet, J.-P., Gallese, V., & Rizzolatti, G. (2003). Both of us disgusted in *my* insula: The common neural basis of seeing and feeling disgust. *Neuron*, **40**, 655–664. doi: 10.1016/S0896-6273(03)00679-2

Williams, G. C. (1996). *Adaptation and natural selection: A critique of some current evolutionary thought*. Princeton, NJ: Princeton University Press.

Woolrich, M. W., Behrens, T. E. J., Beckmann, C. F., Jenkinson, M., & Smith, S. M. (2004). Multi-level linear modelling for FMRI group analysis using Bayesian inference. *NeuroImage*, **21**, 1732–1747. doi: 10.1016/j.neuroimage.2003.12.023

Woolrich, M. W., Jbabdi, S., Patenaude, B., Chappell, M., Makni, S., Behrens, T., et al. (2009). Bayesian analysis of neuroimaging data in FSL. *NeuroImage*, **45**, 173–186. doi: 10.1016/j.neuroimage.2008.10.055

Woolrich, M. W., Ripley, B. D., Brady, J. M., & Smith, S. M. (2001). Temporal autocorrelation in univariate linear modelling of FMRI data. *NeuroImage*, **14**, 1370–1386. doi: 10.1006/nimg.2001.0931

Worsley, K. J. (2001). Statistical analysis of activation images. In P. Jezzard, P. M. Matthews, & S. M. Smith (Eds.), *Functional MRI: An introduction to methods* (pp. 251–270). Oxford, England: Oxford University Press.